Atherosclerosis (PAD + carotid stenosis) Acute Limb Ischemia

Objectives:

Review **of histo-anatomy of a blood vessel.**	• layers of a blood vessel.
Pathophysiology of atherosclerosis.	• definition of atherosclerosis. • hemodynamic facts. • risk factors of atherosclerosis. • steps of atherosclerosis.
Peripheral ischemia.	• Definition. • signs & symptoms of acute ischemia and chronic ischemia • anatomical landmarks of peripheral pulses.
Carotid artery disease.	• anatomy. • risk factors. • history taking. • definition of TIA.

Resources:

- Davidson's.
- Slides
- Surgical recall.
- Raslan's notes.
- Team 434

Done by: Nouf Alabdulkarim & Nouf Altuwaijri
Sub-leader: Lina ismael
Leaders: Abdulrahman Alsayyari & Monerah Alsalouli
Reviewed by: Ahmed Al Yahya & Helmi Alsweirki

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Once you stop learning you start dying.

Basic review:

Vascular system:

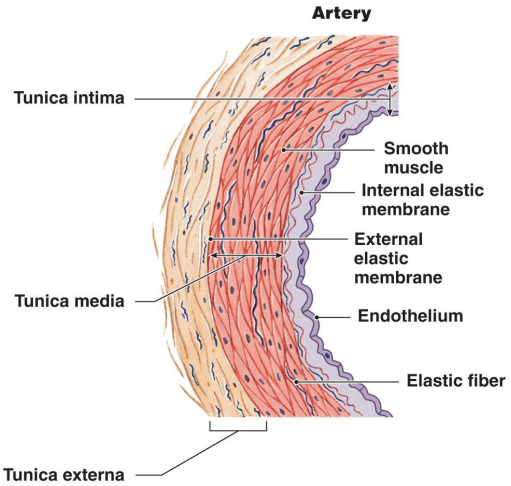
- **Arteries**
- **Capillaries**
- **Veins**

Type of arteries:

Elastic arteries	Muscular arteries	Small arteries
Aorta & Beginning of its large branches (majority of elastic fibers in media)	medium sized arteries, distributing arteries. (exhibit smooth muscles in their walls)	major site of autonomic regulation of blood flow.

Layers of the blood vessel (from inner to outer):

1. **Tunica intima:**
   - endothelial cells
   - internal elastic membrane.
2. **Tunica Media:**
   - smooth muscle fibres
   - external elastic membrane.
3. **Tunica Adventitia.**



What is the function of the Endothelial cells? (hemodynamic facts)

- The endothelial cells that line blood vessels provide an active, dynamic interface between the blood stream and the arterial wall.
- They provide a **semi-permeable barrier** that regulates the exchange of fluid, nutrients, gases, and waste between the blood and tissues. It also transport a little bit of blood protein by pinocytosis[1].
- They provide unique surface that generally allows the cellular elements of blood to flow with adhering to the vessel lining .
- Endothelial cells also regulate the **constriction** and **relaxation** of vessels to regulate the blood flow by releasing:
  - **Vasodilatory molecules:** nitric oxide (NO) and prostacyclin (PGI2)
  - **Vasoconstrictive molecules:** endothelin and angiotensin-II
- **When injury occurs**, endothelial cells secrete **cytokines** that trigger and maintain an **inflammatory response.** "the inflammatory response is the basic **first step** of the atherosclrotic process

[1] the ingestion of liquid into a cell by the budding of small vesicles from the cell membrane.

Atherosclerosis

Introduction:

- Atherosclerosis is the number one killer worldwide and here in Saudi Arabia.
- It's an **inflammatory process** that causes clogging, narrowing and hardening of the **large** and **medium** sized arteries, it's a progressive process throughout the life.
- The process of atherosclerosis starts Since childhood (2 years old) fat streaks start to appear in the blood vessels.
- In prone individuals, risk factors will lead to more accumulation of these fat streaks leading to full blown atherosclerosis and its complications.
- In order to cause a significant drop of the arterial flow at rest the atheromatous plaque must reduce the artery cross sectional area about **70%**

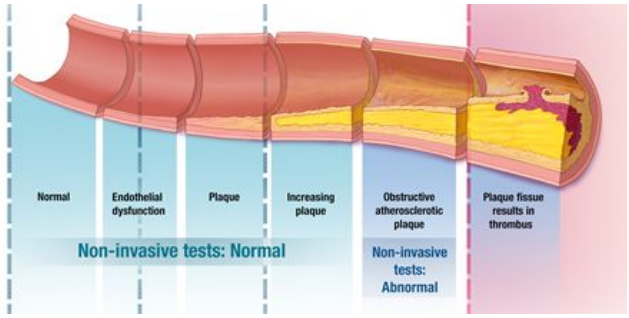
Atherosclerosis accounts **approximately 90%** of patients presenting to vascular specialty, other **less** common causes:

- Aneurysms (hereditary or acquired)
- Trauma/Radiation
- Infection (when they're injecting the drug for example they might harm the vessels or mycotic aneurysms[2])
- Functional Spasms (Raynaud's syndrome)[3]
- Vasculitis
- Anatomic abnormalities (thoracic outlet syndrome)[4]

Endothelial injury Causes : imp

Endothelial injury usually occurs early in life and may be initiated by a chemical or physical injury.

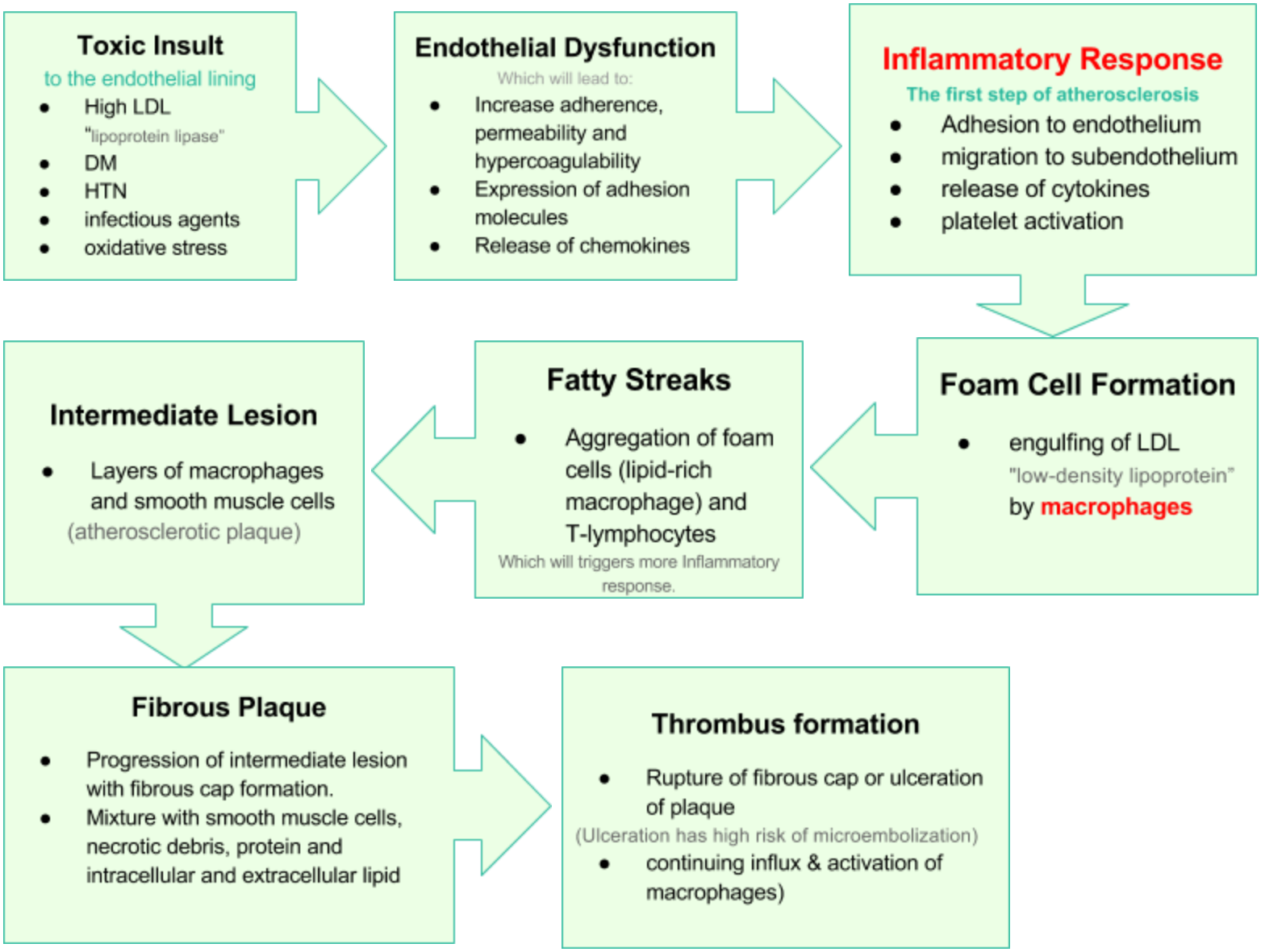
- Physical injury or stress as a result of direct trauma or hypertension. (when high pressure flows into the blood vessel it injures the endothelial lining and therefore the atherosclerotic process starts)
- Turbulent[5] blood flow, for example, where arteries branch. Any bifurcation ex:Aorta.
- Circulation of reactive oxygen species (free radicals), e.g., from smoking or air pollutants.
- Hyperlipidemia (high blood concentrations of LDL or VLDL).
- Chronically elevated blood glucose levels and high carbohydrate intake. (DM)
- **Homocysteinemia**, which results from an inherited metabolic defect that leads to very high levels of the homocysteine, a metabolite of methionine; high concentrations are toxic to the endothelium.



[2] An aneurysm arising from bacterial infection of the arterial wall. It can be a common complication of the hematogenous spread of bacterial infection.
[3] Could be primary (no cause) or secondary (autoimmune), causes some areas of your body — such as your fingers and toes — to feel numb and cold in response to cold temperatures or stress. In **Raynaud's disease**, smaller arteries that supply blood to your skin narrow, limiting blood circulation to affected areas (vasospasm).
[4] a group of disorders that occur when blood vessels or nerves in the space between your collarbone and your first rib (**thoracic outlet**) are compressed. This can cause **pain** in your shoulders and neck and numbness in your fingers.
[5] تموّج

Pathophysiology:

Atherosclerosis is a disease process which is triggered by sometimes subtle physical or chemical insults to the endothelial cell layer of arteries.



the plaque at first is soft and that's dangerous since it might detach and send a micro emboli or the whole plaque detaches and occludes a main blood vessel, but when the plaque reaches the fibrous form it becomes more stable but still dangerous since it might rupture and ulcerate the vessel that creates a tough surface, so when blood flow through it the platelets would gather to create micro embolization, also the plaque by itself might occlude the vessel.

Didn't understand? Check this video!

Risk factors : imp

Modifiable		Non-modifiable
Major	**Minor**	Male
Smoking	Homocystenemia[6]	Age
Hypertension	Hypercoagulable states	After menopause in female
Diabetes Causes very early atherosclerotic changes	Obesity and high carb intake	Family predisposition
	Physical inactivity/ sedentary lifestyle	Genetic abnormality
Hyperlipidemia	Stressful & type A personality[7]	

Complications:

- Disruption of laminar flow
- Ischemia.
- Thrombus formation.

Clinically silent until complications emerge.

Prevention and Treatment:

- **Primary prevention** for modifiable risk factors like lifestyle (in general, we control the risk factors)
- **Secondary prevention** (medications)
    - **Statins** to decrease cholesterol levels.(anti-inflammatory, decrease LDL)
    - **Low dose Aspirin** to minimize the risk of thrombus formation (any antiplatelet medication, not only aspirin).
    - Beta blockers, ACE-I, and Angiotensin receptor antagonists, prevent the complications of PVD e.g. stroke.
- **Treatment** (in general)
    - Endarterectomy (in some arteries, in localized disease) [8]
    - Percutaneous Angioplasty (minimally invasive, we pass a catheter and dilate the artery {the stenotic segment}) - we should do it when the patient has decompensated heart failure, can't tolerate any anesthesia.
    - Bypass procedures.

[6] Elevated homocysteine
[7] Type A behavior characterized by excessive ambition, aggression, competitiveness, drive, impatience, need for control. It is commonly associated with risk of coronary disease and other stress-related ailments.
[8] The removal of material on the inside of an artery.

Surgical recall:

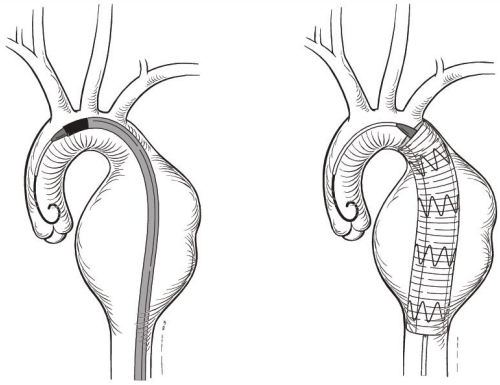
What is atherosclerosis?
Diffuse disease process in arteries; atheromas containing cholesterol and lipid form within the intima and inner media, often accompanied by ulcerations and smooth muscle hyperplasia
What is the common theory of how atherosclerosis is initiated?
Endothelial injury → platelets adhere → growth factors released → smooth muscle hyperplasia/plaque deposition
What are the risk factors for atherosclerosis?
Hypertension, smoking, diabetes mellitus, family history, hypercholesterolemia, high LDL, obesity, and sedentary lifestyle
What are the common sites of plaque formation in arteries?
Branch points (carotid bifurcation), tethered sites (superficial femoral artery [SFA] in Hunter's canal in the leg)
What must be present for a successful arterial bypass operation?

1. Inflow (e.g., patent aorta)
2. Outflow (e.g., open distal popliteal artery)
3. Run off (e.g., patent trifurcation vessels down to the foot)

What is the major principle of safe vascular surgery?
Get proximal and distal control of the vessel to be worked on!
What does it mean to "POTTS" a vessel?
Place a vessel loop twice around a vessel so that if you put tension on the vessel loop, it will occlude the vessel
What is the suture needle orientation through graft versus diseased artery in a graft to artery anastomosis?
Needle "in-to-out" of the lumen in diseased artery to help tack down the plaque and the needle "out-to-in" on the graft
What are the three layers of an artery?

1. Intima
2. Media
3. Adventitia

Which arteries supply the blood vessel itself?
Vaso vasorum
What is a true aneurysm?
Dilation (>2 × nL diameter) of all three layers of a vessel
What is a false aneurysm (a.k.a pseudoaneurysm)?
Dilation of artery not involving all three layers (e.g., hematoma with brous covering) Often connects with vessel lumen and blood swirls inside the false aneurysm
What is "ENDOVASCULAR" repair?
Placement of a catheter in artery and then deployment of a graft intraluminally



Peripheral Arterial Disease (PAD)

It is the sequence of atherosclerosis of peripheral vessels excluding the carotids and coronaries arteries.
Patients with PAD have a 6-fold increased risk of cardiovascular disease mortality compared to patients without PAD, even the patient with or without symptoms.
PAD presents as:

Acute arterial disease:

Acute limb ischaemia is caused most frequently by acute thrombotic **occlusion** of a pre-existing stenotic arterial segment (60%), thromboembolism (30%) and trauma, Embolic got initiated away, thrombotic is local.

Embolic most common	Thrombotic	Traumatic
an obstruction in a blood vessel due to a blood clot or other foreign matter that gets stuck while traveling through the bloodstream. **Sources of an Embolus:** **- Cardiac source most common 80%** Arrhythmias (A-fib), MI, prosthetic valve, endocarditis **- Non-Cardiac 10%:** Proximal plaque, aneurysm, paradoxical emboli [9] **- Iatrogenic 10%:** Angiographic manipulation, Surgical manipulation **Common sites of embolus:** Femoral artery	The local formation or presence of a blood clot in a blood vessel. Thrombosis in situ may arise from acute plaque rupture, hypovolaemia, increased blood coagulability (for example in association with sepsis) or 'pump failure' (for example heart attack)	The most common causes of injury are limb fractures, dislocations, blunt injuries, road traffic accidents, and stab wounds At levels of arteries complete cut causes **vasoconstriction** while partial cut causes **vasodilatation**, so the complete cut is better.

Signs and symptoms: the 6P's very imp

- **Pain**
- **Paralysis**
- **Pallor**
- **Parethesia**
- **Poikilothermia**[10]
- **Pulselessness**

Diagnosis by:

- **Angiogram w/ contrast is the (Golden standard in evaluation)**
- **ECG (looking for MI and atrial fibrillation)**
- **Echocardiogram (looking for clot,MI and valve vegetation)**

Immediate management:

1- IV heparin and anticoagulant **2- angiogram**

Treatment: Surgical embolectomy

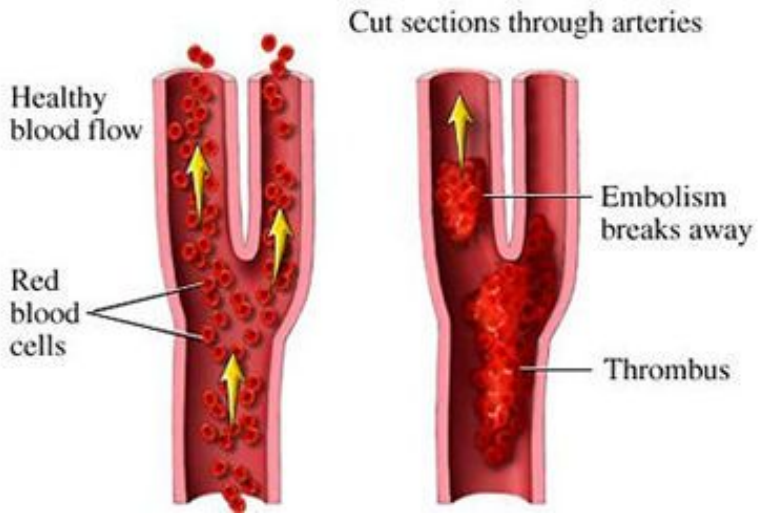
Complication: Gangrene if not treated. (which may lead to amputation)

[9] also called a crossed embolism, refers to an embolus which is carried from the venous circulation to the arterial through a shunt i.e (ASD)
[10] Poikilothermia: impaired regulation of body temperature of the limb usually cool, reflecting the ambient temperature.

Acute arterial disease: cont..

How to differentiate between thrombotic and embolic causes?

thrombosis	embolism
Hx of previous claudication	No hx of arterial insufficiency
No source of emboli	Previous source of emboli (A-fib,MI)
Long history (weeks)	Short history (hrs- days) Sudden onset
Less severe ischemia	More severe ischemia
Lack of pulse in the contralateral leg	present pulse in the contralateral leg
Collaterals are seen on A-gram	Lack of collaterals



Recall:

What is acute arterial occlusion?
Acute occlusion of an artery, usually by embolization; other causes include acute thrombosis of an atheromatous lesion, vascular trauma
What are the classic signs/ symptoms of acute arterial occlusion? The "six P's":

- **P**ain
- **P**aralysis
- **P**allor
- **P**aresthesia
- **P**olar (some say Poikilothermia - you pick)
- **P**ulselessness

What is the classic timing of pain with acute arterial occlusion from an embolus?
Acute onset; the patient can classically tell you exactly when and where it happened
What is the immediate preoperative management?

1. Anticoagulate with IV heparin (bolus followed by constant infusion)
2. A-gram

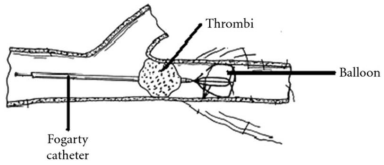
What are the sources of emboli?

1. Heart - 85% (e.g., clot from AFib, clot forming on dead muscle after MI, endocarditis, myxoma)
2. Aneurysms
3. Atheromatous plaque (atheroembolism)

What is the most common cause of embolus from the heart? **A-Fib**
What is the most common site of arterial occlusion by an embolus?
Common femoral artery (SFA is the most common site of arterial occlusion from atherosclerosis)
What diagnostic studies are in order?

1. A-gram
2. ECG (looking for MI, AFib)
3. Echocardiogram (looking for clot, MI, valve vegetation)

What is the treatment?
Surgical embolectomy via cutdown and Fogarty balloon (bypass is reserved for embolectomy failure)
What is a Fogarty?
Fogarty balloon catheter - catheter with a balloon tip that can be inflated with saline; used for embolectomy



What must be looked for postoperatively after reperfusion of a limb?
Compartment syndrome, hyperkalemia, renal failure from myoglobinuria, MI
What is compartment syndrome?
Leg (calf) is separated into compartments by very unyielding fascia; **tissue swelling** from reperfusion can increase the intracompartmental pressure, resulting in decreased capillary flow, ischemia, and myonecrosis; myonecrosis may occur after the intracompartment pressure reaches only 30 mm Hg
What are the signs/ symptoms of compartment syndrome?
Classic signs include pain, especially after passive flexion/extension of the foot, paralysis, paresthesias, and pallor; **pulses are present** in most cases because systolic pressure is much higher than the minimal 30 mmHg needed for the syndrome!
Can a patient have a pulse and compartment syndrome? **YES!**
How is the diagnosis made?
History/suspicion, compartment pressure measurement
What is the treatment of compartment syndrome?
Treatment includes opening compartments via bilateral calf-incision fasciotomies of all four compartments in the calf

Chronic arterial disease:

IC (intermittent claudication)	CLI (chronic limb ischemia)
IC is usually characterized by pain on walking in the muscles of one or both calves. The pain comes on after a reasonably constant 'claudication distance' usually subsides rapidly and completely on cessation of walking. Resumption of walking causes the pain to return. IC affects up to 5% of people aged over 60 years. The pain will occur after walking a fixed distance, and the pain will be relieved after a fixed period of rest. **Common site:** In the lower extremities in the calf muscle, but can occur everywhere. **Classic presentation:** **cramp like pain** caused by walking specific distance and resolved by stopping specific amount of time. **Management:** By conservative treatment (**PACE**): • **P**entoxifylline • **A**spirin. • **C**essation of smoking • **E**xercise	Whereas IC is usually due to single-level disease, CLI is caused by multiple lesions affecting different arterial segments down the leg These patients usually have • Rest pain.(night) pain • Tissue loss in form of (ulceration or gangrene) • Low ABPI[11] Without revascularization, such patients will often lose their limb, and sometimes their life, in a matter of months. Tissue loss and rest pain similar to msk but the difference msk is relieved by analgesia. But CVS not relieved even by 2 weeks of analgesia. **Common site:** dorsalis pedis artery (on the dorsum of the foot above the metatarsal) classically at night awakening the patient. The rest pain can be resolved by standing or hanging the foot on the other side of bed due to gravity that afford more blood flow to ischemic area. **Common sites of ulcer:** Can occur anywhere, but the **toes** and **foot** are the most common. **Treatment:** (surgery intervention) • surgical graft bypass • Angioplasty (balloon dilation) • Endarterectomy (remove diseased intima and media) • surgical patch angioplasty (place patch over stenosis)

Signs and symptoms:

- scaly,dry skin
- hair loss
- muscle atrophy
- absent pulse
- thick toenails
- Buritis
- Ulcer and tissue necrosis

Indication of surgery: (STIR)

- **S**evere claudication
- **T**issue necrosis
- **I**nfection
- **R**est pain.

Diagnosis by : Contrast angiogram (GOLD standard)[12]

[11] The severity of ischaemia in the leg can be simply estimated by determining the ratio between the ankle and brachial blood pressures.
[12] MCQ: What is the gold standard for PAD? Contrast angiography.

PAD risk factors:

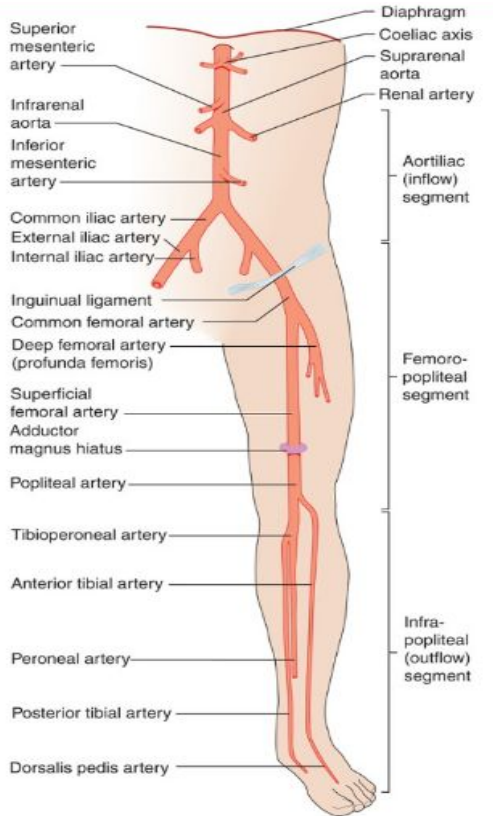
- Hypertension.
- Diabetes.
- Hyperlipidemia.
- Smoking.
- Familial tendency.
- Obesity.
- Gender.

History:

- Pain
  - location.
  - precipitating and aggravating factors.
  - frequency and duration.
- Rule out other causes of pain of lower limb.
- Patients with comorbid conditions and can not walk, present late with rest pain or gangrene.
- Drug/medical history. Ex: 60 y.o patient with big toe gangrene hyperlipidemic, hypertensive and not complied to his medications. Other patient same age same disease but complied to his medications. Of course the patient who is compiled is better. So drug history is very important to know.
- Surgical history.Complications or allergies
- Family history: first degree relative with abdominal aortic aneurysm. Very important

Pain location and site of occlusion:

Pain location	Site of occlusion
Buttock and hip 30%	Aortoiliac disease "**Leriche's syndrome triad":** Bilateral claudication + absent femoral pulses +/- erectile dysfunction (impotence)
Thigh	Aortoiliac or common femoral artery[13]
Upper ⅔ of the calf 60% **(most common)**	Superficial femoral artery
Lower ⅓ of the calf	Popliteal artery
Foot claudication	Tibial arteries (especially in **DM**)



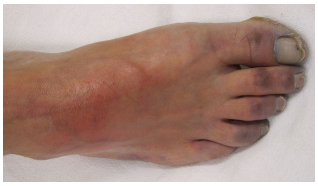
Example: if the abdominal aorta is affected, we will have bilateral intermittent claudication in the lower limbs and there is no blood supply in the pelvis, hence if the patient was a male, he will be impotent.

[13]
- After inguinal ligament >> Common femoral artery
- Before inguinal ligament >> External iliac

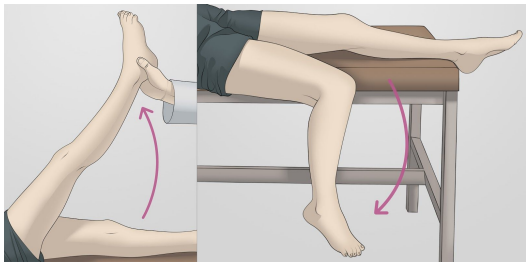
Review of vascular symptoms:

- Transient ischemic attack (TIA) It's a neurological deficit which resolves after few hours. Ask: did you ever have paresthesia and/or loss of vision for a while?
- Difficulty in speech or swallowing
- Dizziness / drop attacks
- Blurred vision (transient loss of vision known as amaurosis fugax)
- Arm fatigue
- Pain in abdomen after eating (known as intestinal angina after 15-20 min of eating the pt will have sever pain due to increased intestinal blood demand after eating and there is already atherosclerosis, so no enough blood supply) In case of **mesenteric** ischemia
- Renal insufficiency (poorly controlled DM+/- HTN) After 15 years of DM complications start to appear but it differs according to if it's controlled or not.
- Impotence (aortoiliac gives the gonadal arteries,so whenever atherscloritic it will decrese blood to the penis so impotence occurs)
- Claudication/rest pain/tissue loss It's better if the patient is claudicant than has rest pain or tissue loss because then we don't have enough options

Physical examination: always **compare** between the limbs.



- **Inspection:**
  - Change in color.Purple then darker to black, often mixed with cellulitis.
  - Signs of ischemia.Signs of chronic ischemia: dry scaly skin because sebaceous glands are affected, Brittle nails , loss of hair and fungal infection in between toes.
  - Burger's test (pale sole).when normal people lay down and raise their legs there will be no change of color, but when ischemic patients raise their legs 20-30 degrees it'll be pale.



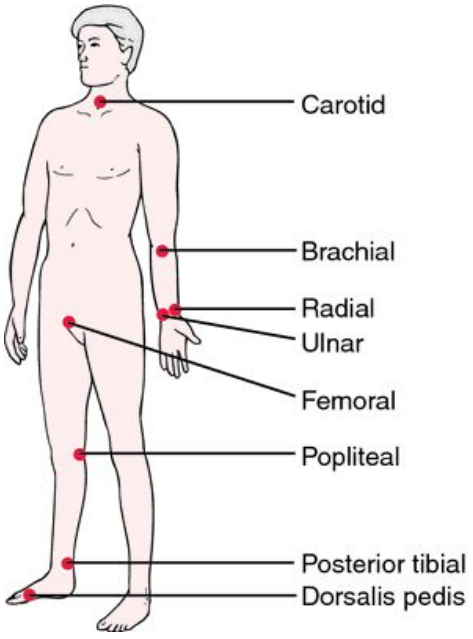
  - Capillary filling. Normally it takes one to two seconds (1-2)
  - Venous refilling (venous guttering).After raising the legs, the patient is asked to dangle them and they're observed. Delayed venous return happens when there is no adequate arterial supply, normally venous return takes about 10 seconds, if it's longer that means it's delayed.
  - Pre Gangrenous /gangrenous part examination. fungal infection, ulcers in pressure areas.
  - Dry scaly skin (atrophic changes, even the sebaceous gland atrophied)

Atrophy of the calf muscles, loss of extremity hair, and thickened toenails are clues to underlying PAD

- **Palpation:**
  - Skin temperature.
  - Venous refilling.
  - Peripheral pulses.

Anatomical landmarks for pulses in lower extremity: imp

Femoral artery	Just below the inguinal ligament
Popliteal artery	at the middle of popliteal fossa
Posterior tibial artery	The posterior tibial artery pulse can be readily palpated halfway between the posterior border of the medial malleolus and the Achilles tendon
Dorsalis pedis artery	the pulse is felt on the top of the foot, between the first and second metatarsal bones.



- **Palpation:** cont...
  - Joint movements /muscle strength.
  - Sensation. Thrill [14](turbulent blood flow felt)

- **Auscultation:**
  - Bruits [15](turbulent blood flow heard)

Palpate the abdominal aorta to the foot, auscultate for bruits in the abdominal and pelvic regions, absence of a pulse signifies arterial obstruction proximal to the area palpated.

Differential diagnosis of claudication:

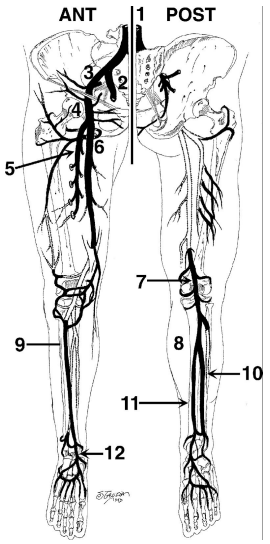
we have to differentiate between claudication resulting from MSK or CVS.

	Arterial	Neurogenic	Venous
Pathology	Stenosis or occlusion of major lower limb arteries	Lumbar nerve roots or cauda equina compression (spinal stenosis)	Obstruction to the venous outflow of the leg due to iliofemoral venous occlusion secondary to deep venous thrombosis
Site of pain	Muscles: usually the calf but may affect thigh and buttock	Ill-defined; whole leg. Shooting in nature; may be associated with tingling and numbness	Whole leg. Bursting in nature
Laterality	Usually unilateral if femoro-popliteal, bilateral if aortoiliac disease	Often bilateral	Nearly always unilateral
Onset	Gradual onset after walking the 'claudication distance'	Often immediate upon walking or even on standing up	Gradual onset but may be present from the moment walking commences
Relieving features	On cessation of walking, the pain disappears completely in 1–2 minutes	On cessation of walking, the pain may gradually subside over 5–10 minutes. Often the patient has to sit down or lean against something to obtain relief	The subject usually needs to elevate the leg to obtain relief
Colour	Normal or pale	Normal	Cyanosed. Often visible varicose veins and venous skin changes
Temperature	Normal or cool	Normal	Normal or increased
Swelling	Absent	Absent	Always present
Pulses	Reduced or absent	Normal	Present, but may be difficult to feel because of swelling
Straight leg raising	Normal	Limited	Normal

[14] Turbulent blood flow that we can **feel** it
[15] Turbulent blood flow that we can **hear** it

Recall:

Define the arterial anatomy:



1.Aorta	2.Internal iliac (hypogastric)	3.External iliac
4.Common femoral artery	5.Profundi femoral artery [16]	6.Superficial femoral artery (SFA)
7.Popliteal artery	8.Trifurcation	9.Anterior tibial artery
10.Peroneal artery	11.Posterior tibial artery	12.Dorsalis pedis artery

How can you remember the orientation of the lower exterior arteries below the knee on A-gram? Use the acronym "**LAMP**":

- **L**ateral **A**nterior tibial
- **M**edial **P**osterior tibial

What is peripheral vascular disease (PVD)?
Occlusive atherosclerotic disease in the lower extremities
What is the most common site of arterial atherosclerotic occlusion in the lower extremities?
Occlusion of the superficial femoral artery SFA in Hunter's canal
What are the symptoms of PVD?
Intermittent claudication, rest pain,erectile dysfunction, sensorimotor impairment and tissue loss
What is intermittent claudication?
Pain, cramping, or both of the lower extremity, usually the calf muscle, after walking a specific distance; then the pain/ cramping resolves after stopping for a specific amount of time while standing; this pattern is reproducible
What is rest pain?
Pain in the foot, usually over the distal metatarsals; this pain arises at rest (classically at night, awakening the patient)
What classically resolves rest pain?
Hanging the foot **over** the side of the bed or standing; gravity affords some extra flow to the ischemic areas
How can vascular causes of claudication be differentiated from nonvascular causes, such as neurogenic claudication or arthritis?
History (in the vast majority of patients) and noninvasive tests; remember, vascular claudication appears after a specific distance and resolves after a specific time of rest while standing (not so with most other forms of claudication)
What is the differential diagnosis of lower extremity claudication?
Neurogenic (e.g., nerve entrapment/discs), arthritis, coarctation of the aorta, popliteal artery syndrome, chronic compartment syndrome, neuromas, anemia, diabetic neuropathy pain
What are the signs of PVD?
Absent pulses, bruits, muscular atrophy, decreased hair growth, thick toenails, tissue necrosis/ulcers/infection
What is the site of a PVD ulcer vs. a venous stasis ulcer?
PVD arterial insu ciency ulcer >> usually on the toes/foot
Venous stasis ulcer >> medial malleolus (ankle)
What is the ABI?
Ankle to **B**rachial **I**ndex (**ABI**); simply, the ratio of the systolic blood pressure at the ankle to the systolic blood pressure at the arm (brachial artery) A:B; ankle pressure taken with Doppler; the ABI is noninvasive
What ABIs are associated with normals, claudicators, and rest pain?

- Normal ABI ≥ 1.0
- Claudicator ABI < 0.6
- Rest pain ABI < 0.4

Who gets false ABI readings?
Patients with calcified arteries, especially those with diabetes

[16] deep femoral artery

What are PVRs?

Pulse **V**olume **R**ecordings; pulse waveforms are recorded from lower extremities representing volume of blood per heartbeat at sequential sites down leg, Large wave form means good collateral blood flow (Noninvasive using pressure cuffs) Prior to surgery for chronic PVD

what diagnostic test will every patient receive?

A-gram (arteriogram: dye in vessel and x-rays) maps disease and allows for best treatment option (i.e., angioplasty vs. surgical bypass vs. endarterectomy) Gold standard for diagnosing PVD

What is the bedside management of a patient with PVD?

1. Sheep skin (easy on the heels)
2. Foot cradle (keeps sheets/blankets of the feet)
3. Skin lotion to avoid further cracks in the skin that can go on to form a fissure and then an ulcer

What are the indications for surgical treatment in PVD? Use the acronym "**STIR**":

- **S**evere claudication refractory to conservative treatment that a ects quality of life/livelihood (e.g., can't work because of the claudication)
- **T**issue necrosis
- **I**nfection
- **R**est pain

What is the treatment of claudication?

For the vast majority, conservative treatment, including exercise, smoking cessation, treatment of H N, diet, aspirin, with or without rental (pentoxifylline)

How can the medical conservative treatment for claudication be remembered? Use the acronym "**PACE**":

- **P**entoxifylline
- **A**spirin
- **C**essation of smoking
- **E**xercise

What is the risk of limb loss with claudication?

5% limb loss at 5 years (Think: 5 in 5), 10% at 10 years (Think: 10 in 10)

What is the risk of limb loss with rest pain?

>50% of patients will have amputation of the limb at some point

In the patient with PVD, what is the main postoperative concern?

Cardiac status, because most patients with PVD have coronary artery disease; ≈20% have an AAA, MI is the most common cause of postoperative death after a PVD operation

What is Leriche's syndrome?

Buttock **C**laudication, **I**mpotence (erectile dysfunction), and leg muscle **A**trophy from occlusive disease of the iliacs/distal aorta **Think: CIA spy Leriche**

- **C**laudication
- **I**mpotence
- **A**trophy

What are the treatment options for severe PVD?

1. Surgical graft bypass
2. Angioplasty - balloon dilation
3. Endarterectomy - remove diseased intima and media
4. Surgical patch angioplasty (place patch over stenosis)

What is DRY gangrene?

Dry necrosis of tissue without signs of infection ("mummified tissue")

What is WET gangrene?

Moist necrotic tissue with signs of infection

What is blue toe syndrome?

Intermittent painful blue toes (or fingers) due to microemboli from a proximal arterial plaque

What are the indications of amputation?

Irreversible tissue ischemia (no hope for revascularization bypass) and necrotic tissue, severe infection, severe pain with no bypassable vessels, or if patient is not interested in a bypass procedure

Carotid Artery Disease

Up to 50% of all ischaemic strokes may be caused by atheroembolism from the carotid bifurcation. Patients with carotid territory transient ischaemic attacks (TIA) and amaurosis fugax (painless temporary loss of vision) **should be assessed by a vascular surgeon with a view to carotid endarterectomy (CEA)**

Risk factors:

- Hypertension.
- Diabetes.
- Hyperlipidemia.
- Smoking.
- Familial tendency.
- Obesity.
- Gender.

History:

- **Transient ischemic attack (TIA):** Loss of motor or sensory function for less than 24 hour
- **Amaurosis Fugax:** transient visual loss for less than 24 hours of Stroke
- Difficulty in speech or swallowing
- Dizziness / drop attacks
- Blurred vision
- Arm fatigue
- Pain in abdomen after eating
- Renal insufficiency (poorly controlled DM+/- HTN)
- Impotence
- Claudication/rest pain/tissue loss

Signs and symptoms:

According to the affected area:

- Bruit (the only sign found in asymptomatic pt)
- Transient Ischemic Attack (**TIA**)
- stroke.

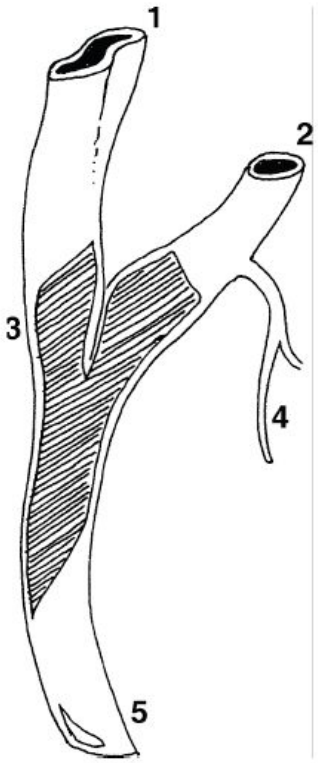
Treatment:

- Goals of treatment:
  1. Prevention of strokes
  2. Prolong survival

Recall:

Identify the following structures:

1. Internal carotid artery
2. External carotid artery
3. Carotid "bulb"
4. Superior thyroid artery
5. Common carotid artery



(Shaded area: common site of plaque formation)

What are the signs/symptoms of carotid vascular disease?

Amaurosis fugax,TIA, RIND and CVA

Define the following terms:

- **Amaurosis fugax:** temporary monocular blindness ("curtain coming down"): seen with microemboli to retina; example of TIA
- **TIA: T**ransient **I**schemic **A**ttack: focal neurologic deficit with resolution of all symptoms within 24 hours
- **RIND: R**eversible **I**schemic **N**eurologic **D**eficit**:** transient neurologic impairment (without any lasting sequelae) lasting 24 to 72 hours
- **CVA: C**erebro**V**ascular **A**ccident (stroke): neurologic deficit with permanent brain damage

What is the risk of a CVA in patients with TIA? ≈10% a year

What is the noninvasive method of evaluating carotid disease?

Carotid ultrasound/Doppler: gives general location and degree of stenosis

What is the gold standard invasive method of evaluating carotid disease? A-gram

What is the surgical treatment of carotid stenosis?

Carotid **E**nd**A**rterectomy (**CEA**): the removal of the diseased intima and media of the carotid artery, often performed with a shunt in place

What are the indications for CEA in the ASYMPTOMATIC patient?

Carotid artery stenosis > 60% (greatest benefit is probably in patients with > 80% stenosis)

What are the indications for CEA in the SYMPTOMATIC (CVA, TIA, RIND) patient?

Carotid stenosis > 50%

Before performing a CEA in the symptomatic patient, what study other than the A-gram should be performed?

Head CT

In bilateral high-grade carotid stenosis, on which side should the CEA be performed in the asymptomatic, right-handed patient?

Left CEA first, to protect the dominant hemisphere and speech center

What is the dreaded complication after a CEA? Stroke (CVA)

What are the possible postoperative complications after a CEA?

CVA, MI, hematoma, wound infection, hemorrhage, hypotension/hypertension, thrombosis, vagus nerve injury (change in voice), hypoglossal nerve injury (tongue deviation toward side of injury-"wheelbarrow" effect), intracranial hemorrhage

What is the mortality rate after CEA? ≈1%

What is the perioperative stroke rate after CEA?

Between 1% (asymptomatic patient) and 5% (symptomatic patient)

What is the postoperative medication?

Aspirin (inhibits platelets by inhibiting cyclo-oxygenase)

What is the most common cause of death during the early postoperative period after CEA? MI

Define "Hollenhorst plaque"?

Microemboli to retinal arterioles seen as bright defects